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Editorial Comment

Management of Sustained Ventricular Tachycardia*

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During recent years, treatment of ventricular tachycardia has attained increasingly higher levels of sophistication. An array of new and more effective antiarrhythmic medications has become available and acute drug testing in the electrophysiology laboratory (1) is rapidly replacing the traditional empirical approach. Similarly, innovative procedures of endocardial resection (2), encircling ventriculotomy (3) and fulguration techniques (4) have been developed, aimed at the eradication of arrhythmogenic foci. Because sustained ventricular tachycardia is potentially a life-threatening arrhythmia, even the most heroic efforts are frequently necessary and justified. In fact, rather than ventricular fibrillation, it is ventricular tachycardia that is progressively being recognized as the initiating arrhythmia in the ongoing epidemic of sudden cardiac death (5,6).

Despite the impressive progress, however, successful management of ventricular tachycardias remains elusive. The reasons for this are numerous. Many patients are refractory to antiarrhythmic medications, and even those who are treated with a drug as effective as amiodarone have a 1 year mortality rate of 10 to 20% (7–9). In addition, the side effects of the various pharmacologic agents can be serious, with noncompliance an ever present possibility. Even more worrisome is the increasing evidence that some of the drugs in use may actually be arrhythmogenic and aggravate rather than prevent the arrhythmia (10,11). With regard to antiarrhythmic surgery, recurrences are still noted in some 20% of patients who survive the operation, and the mortality associated with the procedure itself is approximately 10% (12). This inability to satisfactorily control ventricular tachycardia with pharmacologic and surgical means led to the development of automatic implantable devices aimed at electrically treating arrhythmias (13–15).

In this issue of the Journal, Herre et al. (16) report on 28 patients with recurrent ventricular tachycardia and ventricular fibrillation in whom a pacemaker capable of externally triggered ventricular stimulation was permanently implanted. Twenty-two of these high risk patients were treated with conventional and investigational drugs (group 1), while six underwent mapping-directed endocardial resection (group 2). Although the device was able to provide demand pacing and was used for nonautomatic termination of sustained ventricular tachycardias, its main purpose was to perform serial, noninvasive, electrophysiologic studies.

Role of implanted antitachycardia pacemakers. The study of Herre et al. (16) raises a number of important and provocative questions. The first question deals with the rationale for implanting a permanent device, the primary purpose of which is to obviate repetitive invasive testing. The authors believe that the effort is worthwhile because serial electrophysiologic studies are time-consuming, expensive and, moreover, fraught with complications.

Although these points have merit and the very concept of noninvasive electrophysiologic testing is an attractive one, its chances of becoming broadly accepted could increase markedly if the permanently implanted device were also capable of automatically recognizing and treating ventricular tachyarrhythmias. This is not the case with the pacemakers used in the study of Herre et al. (16). Actually, in the present state of the art, no antitachycardia pacemaker is capable of achieving this objective. Such devices are unsafe because they may accelerate the tachycardia or even induce ventricular fibrillation. They are ineffective because they are unable to terminate a significant proportion of the tachycardias and are powerless in the presence of ventricular fibrillation. They are also unreliable because the individual patient's response to this type of treatment is unpredictable.

It is pertinent to inquire, furthermore, about the clinical outcome of patients with episodes of sustained ventricular tachycardia whose pharmacologic regimen is guided by serial, noninvasive, electrophysiologic studies. A review of the authors' data is revealing. During a mean follow-up period of 8.4 months, 9 of the 22 patients in group 1 developed spontaneous sustained ventricular tachycardia or fibrillation, 6 had intolerable side effects and 4 died from arrhythmia-related causes.

Limitations and advantages of antitachycardia devices. Although management of such high risk patients is admittedly difficult, these results are not encouraging. It is becoming increasingly evident that new approaches are needed to overcome the current inability to successfully manage sustained ventricular tachycardias. Although the search for an ideal drug should continue, improved electronic technology shows significant promise. In fact, antitachycardia pacemakers remain, on both theoretical and clinical grounds, an attractive mode of treatment. Despite present limitations,

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they have a number of inherent advantages such as low energy pulses and the absence of patient distress. While the recently developed implantable cardioverter-defibrillator is safe, effective and aimed at a larger patient group, its high energy output is associated with some degree of discomfort (17,18). The high energy discharges, moreover, are not necessary to terminate the majority of the arrhythmias treated.

It appears that the optimal solution might be to combine in a single device the various electrical treatment modes (19). Such an "ideal" device should first attempt to convert the arrhythmia with pacing or low energy cardioversion and only in case of failure or in the event of acceleration introduce the defibrillating pulse. These technologic developments are at hand and, when associated with the ability to perform serial noninvasive electrophysiologic studies, would represent a quantum leap in our ability to manage ventricular tachyarrhythmias.

References

1. Fisher JD, Cohen HL, Mehra R, Altschuler H, Escher DJW, Furman S. Cardiac pacing and pacemakers. II. Serial electrophysiologic-pharmacologic testing for control of recurrent tachyarrhythmias. Am Heart J 1977;93:658–68.
2. Josephson ME, Harken AH, Horowitz LN. Endocardial excision. A new surgical technique for the treatment of recurrent ventricular tachycardia. Circulation 1979;60:1430–9.
3. Guiraudon G, Fontaine F, Frank R, Escande G, Etievent P, Cabrol C. Encircling endocardial ventriculotomy: a new surgical treatment for life-threatening ventricular tachycardia resistant to medical treatment following myocardial infarction. Ann Thorac Surg 1978;26:438–44.
4. Hartzler GO. Electrode catheter ablation of refractory focal ventricular tachycardia. J Am Coll Cardiol 1983;2:1107–13.
5. Josephson ME, Horowitz LN, Spielman SR, Greenspan AM. Electrophysiologic and hemodynamic studies in patients resuscitated from cardiac arrest. Am J Cardiol 1980;46:948–55.
6. Mason JW, Swerdlow CD, Winkle RA, et al. Ventricular tachyarrhythmia induction for drug selection: experience with 311 patients. In: Lucchesi BR, Dingell J, Schwartz RP Jr, eds. Clinical Pharmacology of Antiarrhythmic Therapy. New York: Raven, 1984:229–39.
7. Morady F, Sauve MJ, Malone P, et al. Long-term efficacy and toxicity of high-dose amiodarone therapy for ventricular tachycardia or ventricular fibrillation. Am J Cardiol 1983;52:975–9.
8. Green HL, Graham EL, Werner JA, et al. Toxic and therapeutic effects of amiodarone in the treatment of cardiac arrhythmia. J Am Coll Cardiol 1983;2:1114–28.
9. Peter T, Hamer A, Weiss D, Mandel WJ. Prognosis after sudden cardiac death without associated myocardial infarction: one year follow-up of empiric therapy with amiodarone. Am Heart J 1984;107:209–13.
10. Velebit V, Podrid P, Lown B, Cohen BH, Graboys TB. Aggravation and provocation of ventricular arrhythmias by antiarrhythmic drugs. Circulation 1982;65:886–94.
11. Ruskin JN, McGovern B, Garan H, DiMarco JP, Kelly E. Antiarrhythmic drugs: a possible cause of out-of-hospital cardiac arrest. N Engl J Med 1983;309:1302–6.
12. Miller JM, Kienzle MG, Harken AH, Josephson ME. Subendocardial resection for ventricular tachycardia: predictors of surgical success. Circulation 1984;70:624–31.
13. Fisher JD, Kim SG, Furman S, Matos JA. Role of implantable pacemakers in control of recurrent ventricular tachycardia. Am J Cardiol 1982;49:194–206.
14. Zipes D, Heger JJ, Miles WM, et al. Early experience with an implantable cardioverter. N Engl J Med 1984;311:485–90.
15. Mirowski M, Reid PR, Mower MM, et al. Termination of malignant ventricular arrhythmias with an implanted automatic defibrillator in human beings. N Engl J Med 1980;303:322–4.
16. Herre JM, Griffin JC, Nielsen AP, et al. Permanent triggered antitachycardia pacemakers in the management of recurrent sustained ventricular tachycardia. J Am Coll Cardiol 1985;6:206–12.
17. Mirowski M, Reid PR, Mower MM, et al. Clinical performance of the implantable cardioverter-defibrillator. PACE 1984;7:1345–50.
18. Echt DS, Armstrong K, Schmidt P, Oyer P, Stinson EB, Winkle RA. Clinical experience, complications, and survival in 70 patients with the automatic implantable cardioverter/defibrillator. Circulation 1985;71:289–96.
19. Winkle RA. Electronic control of ventricular tachyarrhythmias: overview and future directions. PACE 1984;7:1375–9.